Amendments to the Specification:

Please replace the paragraph on pages 6 (lines 17-28) -7, (lines 1-3) as follows:

The present invention relates to: a ~~non-human animal~~ mouse model of Guillain-Barré syndrome which can be obtained by immunizing with gangliosides GQ1b a ~~non-human animal~~ mouse whose FcγRIIB-gene function is deficient in its chromosome to develop Guillan-Barre syndrome ("1"); a ~~non-human animal~~ mouse model of Guillain-Barré syndrome, wherein Guillain-Barré syndrome is Fisher syndrome ("2"); the ~~non-human animal~~ mouse model of Guillain-Barré syndrome according to "1" or "2", which develops a peripheral neuropathy wherein paralysis of its tail and hind legs occurs("3"); ~~the non-human animal model of Guillain-Barré syndrome according to any one of "1" to "3", wherein the FcγRIIB-gene-deficient non-human animal is a rodent ("4"); and the non-human animal model of Guillain-Barré syndrome according to "5", wherein the rodent is a mouse ("5")~~.

Please replace the paragraph on page 7 (lines 4-17) as follows:

The present invention further relates to: a screening method of a therapeutic agent for Guillain-Barré syndrome wherein a test substance is administered to the ~~non-human animal~~ mouse model of Guillain-Barré syndrome according to any one of ~~"1" to "5"~~ "1" to "3", to observe and assess the degree of symptoms of Guillain-Barré syndrome in the ~~non-human animal~~ mouse model of the syndrome("6"); a screening method of a therapeutic agent for Guillain-Barré syndrome and/or Fisher syndrome wherein a test substance is administered to the ~~non-human animal~~ mouse model of Guillain-Barré syndrome according to any one of ~~"1" to "5"~~ "1" to "3", to measure and assess the level of anti-GQ1b antibody appearance("7"); and a therapeutic agent that can be obtained by the screening method of a therapeutic agent for Guillain-Barré syndrome according to "6" or "7"("8").

Please replace the paragraph on page 14 (lines 10-21) as follows:

The wild-type mice and FcγRIIB-gene-deficient mice immunized with GQ1b, were scored for an assessment into five levels according to their symptoms: 0 point-no symptom; 1 point-paralysis of the tail; 2 points-paralysis of the tail and both hind legs; 3 points-paralysis of the tail and all four limbs; 4 points-death. The result is shown in Figure 2. Meanwhile in Figure 2, ~~black squares (▪)~~ white squares (▫) represent the scores for the wild-type mice and ~~white squares (▫)~~ black squares (▪) those for FcγRIIB-gene-deficient mice. As a result, FcγRIIB-gene-deficient mice immunized with GQ1b were observed to develop Guillain-Barré syndrome (Fisher syndrome) (Figure 2).

Please replace the paragraph on pages 14-16 (p. 14 lines 23-27; p. 15 lines 1-29; p. 16 line 1) as follows:

In addition, blood samples of wild-type mice and FcγRIIB-gene-deficient mice both immunized with GQ1b were collected from their orbits 3, 6, 9, and 12 weeks after the primary immunization to examine the level of antibody titer against GQ1b by employing the following improved ELISA analysis provided in a literature (Cell. Immunol. 145, 299-310, 1992). 5 μg of GQ1b was dissolved in 1 ml of 50mM sodium bicarbonate solution (pH=8.5) to be used at the rate of 50 μl per well for coating positively-charged 96-well micro plates (NUNC) overnight at 4°C. Subsequently, the plates were washed once with PBS containing 0.05% of Tween 20 and 0.1% of BSA, and left overnight at 4°C in 250 μl of PBS containing 0.5% of BSA per well for blocking. The sera derived from the above blood and diluted by 500 folds were then added to the above 96-well micro plates at the rate of 50 μl per well to be reacted overnight at 4°C. Following the reaction, the 96-well micro plates were washed three times with PBS containing 0.05% of Tween 20, added 50 μl of 500-fold-diluted goat-anti-mouse IgG1, IgG2a or IgG2b binding to peroxidase (Sigma), and incubated for two

hours at 4°C, and after the incubation, were re-washed three times with PBS including 0.05% of Tween20, went through 30-minute enzymatic reaction with 50 μl of True Blue Peroxidase Substrate (Kirkegaard & Perry Labs) at ambient temperature. OD450 was then measured by a micro-plate reader (Biolumin 960; Molecular Dynamics). The result is shown in Figure 3. Meanwhile in Figure 3, ~~black squares (▪)~~ white squares (▫) represent the absorbance of the wild-type mice and ~~white squares (▫)~~ black squares (▪) that of FcγRIIB-gene-deficient mice. These results have shown that FcγRIIB-knockout mice (IIB-KO)displayed more increased level of antibody titer (IgG1, IgG2a, and IgG2b) against GQ1b than the wild-type mice (Wild), which is consistent with the observation of Guillain-Barré syndrome (Fisher syndrome). It was thus found that a model mouse of Guillain-Barré syndrome (Fisher syndrome) can be generated.